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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/771,961	01/29/2001	C. Alexander Turner JR.	LEX-0121-USA	9694

24231 7590 07/15/2003

LEXICON GENETICS INCORPORATED
8800 TECHNOLOGY FOREST PLACE
THE WOODLANDS, TX 77381-1160

EXAMINER	
HAMUD, FOZIA M	

ART UNIT	PAPER NUMBER
1647	19

DATE MAILED: 07/15/2003

Please find below and/or attached an Office communication concerning this application or proceeding.

Advisory Action	Application No.	Applicant(s)	
	09/771,961	TURNER ET AL.	
	Examiner	Art Unit	
	Fozia M Hamud	1647	

--The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

THE REPLY FILED 05 May 2003 FAILS TO PLACE THIS APPLICATION IN CONDITION FOR ALLOWANCE. Therefore, further action by the applicant is required to avoid abandonment of this application. A proper reply to a final rejection under 37 CFR 1.113 may only be either: (1) a timely filed amendment which places the application in condition for allowance; (2) a timely filed Notice of Appeal (with appeal fee); or (3) a timely filed Request for Continued Examination (RCE) in compliance with 37 CFR 1.114.

PERIOD FOR REPLY [check either a) or b)]

a) ☒ The period for reply expires 6 months from the mailing date of the final rejection.

b) ☐ The period for reply expires on: (1) the mailing date of this Advisory Action, or (2) the date set forth in the final rejection, whichever is later. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the mailing date of the final rejection.
ONLY CHECK THIS BOX WHEN THE FIRST REPLY WAS FILED WITHIN TWO MONTHS OF THE FINAL REJECTION. See MPEP 706.07(f).

Extensions of time may be obtained under 37 CFR 1.136(a). The date on which the petition under 37 CFR 1.136(a) and the appropriate extension fee have been filed is the date for purposes of determining the period of extension and the corresponding amount of the fee. The appropriate extension fee under 37 CFR 1.17(a) is calculated from: (1) the expiration date of the shortened statutory period for reply originally set in the final Office action; or (2) as set forth in (b) above, if checked. Any reply received by the Office later than three months after the mailing date of the final rejection, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

1. ☒ A Notice of Appeal was filed on *01 April 2003*. Appellant's Brief must be filed within the period set forth in 37 CFR 1.192(a), or any extension thereof (37 CFR 1.191(d)), to avoid dismissal of the appeal.

2. ☐ The proposed amendment(s) will not be entered because:

   (a) ☐ they raise new issues that would require further consideration and/or search (see NOTE below);

   (b) ☐ they raise the issue of new matter (see Note below);

   (c) ☐ they are not deemed to place the application in better form for appeal by materially reducing or simplifying the issues for appeal; and/or

   (d) ☐ they present additional claims without canceling a corresponding number of finally rejected claims.

   NOTE: _____.

3. ☒ Applicant's reply has overcome the following rejection(s): See Continuation Sheet.

4. ☐ Newly proposed or amended claim(s) _____ would be allowable if submitted in a separate, timely filed amendment canceling the non-allowable claim(s).

5. ☐ The a)☐ affidavit, b)☐ exhibit, or c)☐ request for reconsideration has been considered but does NOT place the application in condition for allowance because: _____.

6. ☐ The affidavit or exhibit will NOT be considered because it is not directed SOLELY to issues which were newly raised by the Examiner in the final rejection.

7. ☒ For purposes of Appeal, the proposed amendment(s) a)☐ will not be entered or b)☒ will be entered and an explanation of how the new or amended claims would be rejected is provided below or appended.

   The status of the claim(s) is (or will be) as follows:

   Claim(s) allowed: _____.

   Claim(s) objected to: _____.

   Claim(s) rejected: *1-8*.

   Claim(s) withdrawn from consideration: _____.

8. ☐ The proposed drawing correction filed on _____ is a)☐ approved or b)☐ disapproved by the Examiner.

9. ☐ Note the attached Information Disclosure Statement(s)(PTO-1449) Paper No(s). _____.

10. ☒ Other: *answer to arguments attached*

Continuation of 3. Applicant's reply has overcome the following rejection(s): The rejection of claim 2, made under 35 U.S.C.§ 112, second paragraph and the rejection of claims 6 and 8 made under 35 U.S.C § 101..

ADVISORY ACTION

1. Receipt of Applicant's arguments and amendment, filed on 05 May 2003 in Paper No.13, is acknowledged. Claim 2 has been amended. Claims 1-8 are pending and under consideration.

2. The following previous rejections and objections are withdrawn in light of Applicants amendments filed in Paper No.13, 05/05/03:

(I) The rejection of claim 2, made under 35 U.S.C.§ 112, second paragraph.

(II) The rejection of claims 6 and 8 made under 35 U.S.C § 101, for reciting "a cell .....", is withdrawn. Applicants' argument that the cell recited in claims 6 and 8 is limited to a cell that comprises the expression vector of claims 5 and 7, and therefore, does not encompass " the cell" as it occurs in nature, is persuasive.

Claim Rejections - 35 U.S.C. § 101/112

35 U.S.C. 101 reads as follows:

> Whoever invents or discovers any new and useful process, machine, manufacture, or composition of matter, or any new and useful improvement thereof, may obtain a patent therefor, subject to the conditions and requirements of this title.

3a. Claims 1-8 stand rejected under 35 U.S.C. § 101, for reasons of record, set forth in the office actions mailed on 07/02/02 in Paper No:8, and on 12/30/02 in Paper NO:10, because the claimed invention is not supported by either a specific and substantial asserted utility or a well established utility.

Applicants present the following arguments asserting that the claimed invention is supported by a specific and substantial asserted utility or a well-established utility.

I. Applicants contend that they have presented credible evidence that the claimed nucleic acid encodes a novel human membrane protein similar to CD82 and that this

family of proteins plays important role as cell surface receptors and mediators of cell-cell interaction and signal transduction. Thus Applicants assert that the skilled artisan would be able to readily identify that the claimed nucleic acid encodes a human membrane protein similar to CD82. Applicants submit that, although the claimed SEQ ID NO:1 and 2 are identical to BCL-X like polypeptide disclosed in WO/200157213-A2, (which is related to the instant application), that Applicants are no longer pursuing that PCT application.

This argument is not found persuasive. In the response filed on 08 October 2002 in Paper NO:9, Applicants asserted that the claimed nucleic acid encodes a protein similar to CD82 antigen, also known as inducible membrane protein R2, C33 antigen, IA4, metastasis Suppressor Kangi and Suppressor Tumorigenicity-6. Applicants presented sequence homology in **Exhibit E** of that response, however, it appears that the sequence presented in IPI00083978.2 has no homology to instant SEQ ID NO:2. Furthermore, Applicants have not indicated the percent homology between the sequences of the instant invention and that of CD82, neither do Applicants disclose how the protein of the instant invention is related to CD82. Therefore, Applicants have not presented any evidence that clearly shows that the claimed nucleic acid encodes a member of CD82 family. Contrary to Applicants' argument, there is a strong evidence that the claimed nucleic acid and the encoded polypeptide are related to BcL protein. As was set forth in the previous office action, Applicants' WO/200157213-A2 discloses Bcl-X like sequences that share 100% to SEQ ID NO:1 and 2, (it is irrelevant whether Applicants are pursing their PCT Application or not). Further evidence that the claimed

invention is related to Bcl proteins is provided by the fact that instant SEQ ID NO:2 shares 100% homology to Bcl-G, a new member of Bcl-2 family, (see sequence comparison, as well as Guo et al, JBC, vol.276, pages 2780-2785, 2001, post filing date publication). Therefore, it appears that the claimed invention is related to Bcl protein family. Even if the claimed nucleic acid and the encoded protein are members of the CD82 family, Applicants have not demonstrated that there is a common biological role for all the members of this family. For instance, is the protein encoded by the claimed nucleic acid a receptor, and if so what are the cognate ligands that bind it? What signal transduciton pathways does it participate?

II. Applicants argue that patentable utility is distinct from, and does not require knowledge of physiological functions and that unless Applicant is claiming a physiological function, evidence of a physiological function is not required to demonstrate patentable utility. Structural claims are sufficiently supported by structural disclosure, as defined by 35 U.S.C. § 101.

This argument has been fully considered but is not deemed persuasive. Applicant attention is directed to 35 U.S.C. §101, which reads as follows:

> Whoever invents or discovers any new and useful process, machine, manufacture, or composition of matter, or any new and useful improvement thereof, may obtain a patent therefor, subject to the conditions and requirements of this title.

According to this statute, the claimed invention must not only be "new", but must also be "useful", thus disclosing only the structure of the claimed sequence, without out disclosing what the structure is "useful" for, would not be sufficient to satisfy this statute.

Furthermore, the physiological significance (which is distinct from physiological function) of the claimed sequence must be disclosed, in order for the claimed sequence to be useful diagnostically or therapeutically.

III. Applicants also argue that the final office action seems to be implying, that the claimed invention lacks utility due to the fact that Applicants sequences are novel.

Applicant is mischaracterizing the examiner's position. The final office action clearly explained that the instant specification does not disclose the biological or physiological significance of the claimed nucleic acid or the encoded polypeptide. A specification can meet the legal requirements of utility and enablement for a new polynucleotide as long as the specification discloses a credible, specific and substantial asserted utility for the new polynucleotide, or a well-established utility for the claimed polynucleotide. For example, if a novel polynucleotide is shown to be expressed in colon cancer and not expressed in healthy colon tissue, but there is no disclosure of the biological activity of the polypeptide encoded by the polynucleotide, said polynucleotide would not be rejected under 35 U.S.C. §§ 101 and 112, first paragraph, as it has utility and is enabled as a colon cancer marker. However, such is not the fact pattern in the instant case.

VI. Applicants argue that knowledge of the exact function or role of the claimed nucleic acid is not required for said nucleic acid to be used to track expression pattern in a DNA gene chip. The claimed sequence provides a specific marker of the human genome which can be used as targets for discovering drugs that are associated with human disease. Thus, Applicants conclude that compositions that enhance the utility of

such DNA chips must also be useful. Applicants also submit since only 2-4% of the genome encodes proteins, therefore, not all human genomic DNA sequence is useful in DNA gene chip application. Applicants assert that an entire industry is established based on the use of gene sequences in gene chip format, and Applicants cites few well known companies such Affymetrix, Gene Logic, ABI-Perkin-Elmer, HySeq and Incyte. Applicant also argues that real world "substantial" utility for the claimed sequence is provided by the fact that Rosetta Inpharmatics, a gene company is acquired by Merck & Co, for substantial sum of money, $620 million. Therefore, Applicants conclude, that a person skilled in the art would recognize the utility, both scientific and commercial of genomic data in general and specifically human genomic data. Also, since human genomic data is worth billions of dollars it must have well-established utility.

This argument is fully considered but is deemed unpersuasive. Using the claimed nucleic acid a chromosomal marker does not provide the claimed invention s specific utility, because no meaningful information will be obtained from tracking the level of expression of the claimed nucleotide, because there is no physiological or biological significance attached to these nucleotides or the encoded proteins. Without a disclosure of a particular disease state in which the claimed polynucleotides are expressed at an altered level or form, it would be impossible to determine what the results of a gene expression monitoring assay mean. For example, if a compound is tested on a microarray comprising the claimed polynucleotides and affects expression of the polynucleotides negatively, it cannot be determined if that means that the compound is a potential good drug for a disease or would acerbate the disease if

administered. The test results also would not have meaning in terms of what specific disease is relevant. The asserted utility in gene expression monitoring assays is thus not substantial, because significant further research would have to be conducted to determine which diseases correlate with altered forms or levels of the claimed polynucleotides, and whether the claimed polynucleotides are overexpressed or underexpressed in the diseased tissue. Furthermore, since any expressed polynucleotide can be added to a microarray for gene expression monitoring, the asserted utility is not specific to the claimed polynucleotides. The specification does not disclose that the claimed gene is a marker for specific diseases. Absent a disclosure of altered levels or forms of a gene in diseased tissue as compared with the corresponding healthy tissue, the gene is not a disease marker or an appropriate target for drug discovery or toxicology testing. The fact that there is an entire multi billion dollar industry on gene chip technology, does not provide the claimed invention with specific or well established utility, because, this revolutionizing technology enables scientists to attain ambitious goals from identifying genetic variations associated with disease to discovering new drug targets, however, instant application is not drawn to a novel gene chip technology, but rather to nucleic acid sequences with no known physiological role. Furthermore, evidence of commercial success, while sometimes persuasive as secondary evidence of non-obviousness, is immaterial to utility and enablement. Many products have enjoyed commercial success due to fads or clever advertising, wherein the products would not have met the legal standards for utility and enablement.

VI Applicants contend that they fail to understand how an invention fully disclosed and free of prior art at the time of filing could less utility and less enabled than inventions in issued patents. Applicants' invention is more enabled and retains at least as much utility as the inventions described in the claims of U.S. Patents of record.

With respect to this argument, Applicants are reminded that each Patent Application is examined on its' own merits and each Patent Application must meet the criteria set forth in the Revised Interim Utility Guidelines, for a specific and substantial credible asserted utility, or a well established utility.

Applicants are referred to the interim guidelines on utility published on December 21, 1999 in the federal Register, volume 64, Number 244 pp 71427-71440. (Also available at www.USPTO.gov). And the training materials also found on the same web site.

3b. The claimed invention also stands rejected under 35 U.S.C. 112, first paragraph, since the claimed invention is not supported by either a specific and substantial asserted utility or a well established utility for the reasons set forth above, because one skilled in the art clearly would not know how to use it.

Conclusion

4. No claim is allowed.

Advisory Information

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Fozia M Hamud whose telephone number is (703) 308-8891. The examiner can normally be reached on Monday, Wednesday-Thursday, 6:30 am to 4:00 pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Gary Kunz can be reached on (703) 308-4623. The fax phone numbers for

the organization where this application or proceeding is assigned are (703) 308-4227 for regular communications and (703) 308-0294 for After Final communications.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to the receptionist whose telephone number is (703) 308-0196.

Fozia Hamud
Patent Examiner
Art Unit 1647
July 13, 2003

GARY KUNZ
SUPERVISORY PATENT EXAMINER
TECHNOLOGY CENTER 1600

GenCore version 4.5

OM protein - protein search, using sw model

Run on: June 19, 2002, 16:33:07 ; Search time 46.53 Seconds
(without alignments)
1215.761 Million cell updates/sec

Title: US-09-771-961-2
Perfect score: 1709
Sequence: 1 MCSTSGCDLEEIPLDDDDLN.....WIQQHGGWEKILGISHEEVD 327

Scoring table: BLOSUM62
Gapop 10.0 , Gapext 0.5

Searched: 562222 seqs, 172994929 residues

Total number of hits satisfying chosen parameters: 562222

Minimum DB seq length: 0
Maximum DB seq length: 2000000000

Post-processing: Minimum Match 0%
Maximum Match 100%
Listing first 45 summaries

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1: sp_archea:*
2: sp_bacteria:*
3: sp_fungi:*
4: sp_human:*
5: sp_invertebrate:*
6: sp_mammal:*
7: sp_mhc:*
8: sp_organelle:*
9: sp_phage:*
10: sp_plant:*
11: sp_rodent:*
12: sp_virus:*
13: sp_vertebrate:*
14: sp_unclassified:*
15: sp_rvirus:*
16: sp_bacteriap:*
17: sp_archeap:*

Pred. No. is the number of results predicted by chance to have a score greater than or equal to the score of the result being printed, and is derived by analysis of the total score distribution.

SUMMARIES

Result No.	Score	% Query Match	Length	DB	ID	Description
1	1709	100.0	327	4	Q9BZR8	Q9bzr8 homo sapien
2	1189.5	69.6	252	4	Q9BZR7	Q9bzr7 homo sapien
3	1182	69.2	276	4	Q96QR5	Q96qr5 homo sapien
4	1082.5	63.3	328	11	Q9CPT0	Q9cpt0 mus musculu
5	1081.5	63.3	328	11	Q9D3W3	Q9d3w3 mus musculu
6	124	7.3	334	4	Q9HB09	Q9hb09 homo sapien
7	121	7.1	250	4	Q96I96	Q96i96 homo sapien
8	119	7.0	233	11	O35844	O35844 mus musculu
9	117	6.8	235	11	O35843	O35843 mus musculu
10	116	6.8	188	11	Q9QWX2	Q9qwx2 mus musculu
11	115	6.7	188	4	Q9H1R6	Q9h1r6 homo sapien
12	115	6.7	217	11	Q99N35	Q99n35 mus musculu
13	115	6.7	233	6	Q9N1A2	Q9n1a2 sus scrofa
14	115	6.7	562	10	Q9LXU9	Q9lxu9 arabidopsis
15	113.5	6.6	386	4	Q9UKN3	Q9ukn3 homo sapien
16	113.5	6.6	1693	3	Q9P655	Q9p655 neurospora
17	113	6.6	219	11	Q99N36	Q99n36 mus musculu
18	111.5	6.5	180	6	Q9BDX7	Q9bdx7 bos taurus
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21	110	6.4	485	4	Q9BXK5	Q9bxk5 homo sapien
22	110	6.4	485	4	Q96IB7	Q96ib7 homo sapien
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24	105.5	6.2	238	13	Q90Z98	Q90z98 brachydanio
25	104	6.1	414	2	Q52259	Q52259 escherichia
26	100	5.9	2052	16	Q97K01	Q97k01 clostridium
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28	97.5	5.7	462	16	Q97NM3	Q97nm3 streptococc
29	95.5	5.6	342	11	Q9CUD8	Q9cud8 mus musculu
30	95.5	5.6	366	11	Q9JMB0	Q9jmb0 mus musculu
31	95.5	5.6	732	10	Q9LIT3	Q9lit3 oryza sativ
32	95.5	5.6	1003	5	Q9VKW4	Q9vkw4 drosophila
33	95.5	5.6	1253	11	Q9EQS8	Q9eqs8 mus musculu
34	94.5	5.5	572	10	Q9SR96	Q9sr96 arabidopsis
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38	93.5	5.5	1409	5	Q9V3A7	Q9v3a7 drosophila
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40	93	5.4	204	13	Q90ZH2	Q90zh2 xenopus lae
41	93	5.4	450	3	O13722	O13722 schizosacch
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44	92.5	5.4	255	11	Q9D3J3	Q9d3j3 mus musculu
45	92.5	5.4	4202	11	Q91V63	Q91v63 mus musculu

ALIGNMENTS

Sequence homology to SEQ ID NO: 2

RESULT 1
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AC Q9BZR8;
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DT 01-JUN-2001 (TrEMBLrel. 17, Last sequence update)
DT 01-DEC-2001 (TrEMBLrel. 19, Last annotation update)
DE APOPTOSIS REGULATOR BCL-G LONG FORM.
GN BCLG.
OS Homo sapiens (Human).
OC Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
OC Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
OX NCBI_TaxID=9606;
RN [1]
RP SEQUENCE FROM N.A.
RX MEDLINE=21264734; PubMed=11054413;
RA Guo B., Godzik A., Reed J.C.;
RT "Bcl-G, a novel pro-apoptotic member of the Bcl-2 family.";
RL J. Biol. Chem. 276:2780-2785(2001).
DR EMBL; AF281254; AAG59793.1; -.
DR InterPro; IPR002475; BCL2_family.
DR InterPro; IPR000712; Bcl_2.
DR Pfam; PF00452; Bcl-2; 1.
DR SMART; SM00337; BCL; 1.
DR PROSITE; PS50062; BCL2_FAMILY; 1.
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Matches 327; Conservative 0; Mismatches 0; Indels 0; Gaps 0;

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SEQ NO 4

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Qy     181 FVTEGLSFQLQGHVPVASSSKKDEEEQILAKIVELLKYSGDQLERKLKKDKALMGHFQDG 240
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Qy     241 LSYSVFKTITDQVLMGVDPRGESEVKAQGFKAALVIDVTAKLTAIDNHPMNRVLGFGTKY 300
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Qy     301 LKENFSPWIQQHGGWEKILGISHEEVD 327
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DT 01-JUN-2001 (TrEMBLrel. 17, Last sequence update)
DT 01-DEC-2001 (TrEMBLrel. 19, Last annotation update)
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GN BCLG.
OS Homo sapiens (Human).
OC Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
OC Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
OX NCBI_TaxID=9606;
RN [1]
RP SEQUENCE FROM N.A.
RX MEDLINE=21264734; PubMed=11054413;
RA Guo B., Godzik A., Reed J.C.;
RT "Bcl-G, a novel pro-apoptotic member of the Bcl-2 family.";
RL J. Biol. Chem. 276:2780-2785(2001).
DR EMBL; AF281255; AAG59794.1; -.
SQ SEQUENCE 252 AA; 28089 MW; 87D2E5123EFCB9E4 CRC64;

Query Match 69.6%; Score 1189.5; DB 4; Length 252;
Best Local Similarity 93.5%; Pred. No. 5.4e-88;
Matches 232; Conservative 2; Mismatches 7; Indels 7; Gaps 1;

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Qy      61 CSANESWTEVSWPCRNSQSSEKAINLGKKKSSWKAFFGVVEKEDSQSTPAKVSAQGQRTL 120
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Qy     121 EYQDSHSQQWSRCLSNVEQCLEHEAVDPKVISIANRVAEIVYSWPPPQATQAGGFKSKEI 180
           ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db     121 EYQDSHSQQWSRCLSNVEQCLEHEAVDPKVISIANRVAEIVYSWPPPQATQAGGFKSKEI 180

Qy     181 FVTEGLSFQLQGHVPVASSSKKDEEEQILAKIVELLKYSGDQLERKLKK-------DKAL 233
           ||||||||||||||||||||||||||||||||||||||||||||||||||       | ::
Db     181 FVTEGLSFQLQGHVPVASSSKKDEEEQILAKIVELLKYSGDQLERKDTAFIPIPLVDTSI 240

Qy     234 MGHFQDGL 241
           |  ||||
Db     241 QGFPQDGL 248
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RESULT 3
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DT 01-DEC-2001 (TrEMBLrel. 19, Created)
DT 01-DEC-2001 (TrEMBLrel. 19, Last sequence update)
DT 01-DEC-2001 (TrEMBLrel. 19, Last annotation update)
DE APOPTOSIS REGULATOR BCL-G MEDIAN FORM.
GN BCLG.
OS Homo sapiens (Human).
OC Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
OC Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
OX NCBI_TaxID=9606;
RN [1]
RP SEQUENCE FROM N.A.
RA Montpetit A., Boily G., Sinnett D.;
RT "A detailed transcriptional map of the chromosome 12p12 tumor
RT suppressor locus.";
RL Submitted (JUN-2001) to the EMBL/GenBank/DDBJ databases.
DR EMBL; AY040274; AAK72109.1; -.
SQ SEQUENCE 276 AA; 30948 MW; 81559A7190F5598E CRC64;

Query Match 69.2%; Score 1182; DB 4; Length 276;
Best Local Similarity 99.1%; Pred. No. 2.5e-87;
Matches 227; Conservative 1; Mismatches 1; Indels 0; Gaps

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Qy       1 MCSTSGCDLEEIPLDDDDLNTIEFKILAYYTRHHVFKSTPALFSPKLLRTRSLSQRGLGN 60
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Db       1 MCSTSGCDLEEIPLDDDDLNTIEFKILAYYTRHHVFKSTPALFSPKLLRTRSLSQRGLGN 60

Qy      61 CSANESWTEVSWPCRNSQSSEKAINLGKKKSSWKAFFGVVEKEDSQSTPAKVSAQGQRTL 120
           ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db      61 CSANESWTEVSWPCRNSQSSEKAINLGKKKSSWKAFFGVVEKEDSQSTPAKVSAQGQRTL 120

Qy     121 EYQDSHSQQWSRCLSNVEQCLEHEAVDPKVISIANRVAEIVYSWPPPQATQAGGFKSKEI 180
           ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db     121 EYQDSHSQQWSRCLSNVEQCLEHEAVDPKVISIANRVAEIVYSWPPPQATQAGGFKSKEI 180

Qy     181 FVTEGLSFQLQGHVPVASSSKKDEEEQILAKIVELLKYSGDQLERKLKK 229
           ||||||||||||||||||||||||||||||||||||||||||||||||| |:
Db     181 FVTEGLSFQLQGHVPVASSSKKDEEEQILAKIVELLKYSGDQLERKEKE 229
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RESULT 4
Q9CPT0
ID Q9CPT0 PRELIMINARY; PRT; 328 AA.
AC Q9CPT0;
DT 01-JUN-2001 (TrEMBLrel. 17, Created)
DT 01-JUN-2001 (TrEMBLrel. 17, Last sequence update)
DT 01-JUN-2001 (TrEMBLrel. 17, Last annotation update)
DE 9030625M01RIK PROTEIN (4933405K19RIK PROTEIN).
GN 9030625M01RIK OR 4933405K19RIK.
OS Mus musculus (Mouse).
OC Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
OC Mammalia; Eutheria; Rodentia; Sciurognathi; Muridae; Murinae; Mus.
OX NCBI_TaxID=10090;
RN [1]
RP SEQUENCE FROM N.A.
RC STRAIN=C57BL/6J; TISSUE=COLON, STOMACH, AND TESTIS;
RX MEDLINE=21085660; PubMed=11217851;
RA Kawai J., Shinagawa A., Shibata K., Yoshino M., Itoh M., Ishii Y.,
RA Arakawa T., Hara A., Fukunishi Y., Konno H., Adachi J., Fukuda S.,
RA Aizawa K., Izawa M., Nishi K., Kiyosawa H., Kondo S., Yamanaka I.,
RA Saito T., Okazaki Y., Gojobori T., Bono H., Kasukawa T., Saito R.,
RA Kadota K., Matsuda H.A., Ashburner M., Batalov S., Casavant T.,
RA Fleischmann W., Gaasterland T., Gissi C., King B., Kochiwa H.,
RA Kuehl P., Lewis S., Matsuo Y., Nikaido I., Pesole G., Quackenbush J.,
RA Schriml L.M., Staubli F., Suzuki R., Tomita M., Wagner L., Washio T.,
RA Sakai K., Okido T., Furuno M., Aono H., Baldarelli R., Barsh G.,
RA Blake J., Boffelli D., Bojunga N., Carninci P., de Bonaldo M.F.,
RA Brownstein M.J., Bult C., Fletcher C., Fujita M., Gariboldi M.,
RA Gustincich S., Hill D., Hofmann M., Hume D.A., Kamiya M., Lee N.H.,
RA Lyons P., Marchionni L., Mashima J., Mazzarelli J., Mombaerts P.,
RA Nordone P., Ring B., Ringwald M., Rodriguez I., Sakamoto N.,
RA Sasaki H., Sato K., Schoenbach C., Seya T., Shibata Y., Storch K.-F.,
RA Suzuki H., Toyo-oka K., Wang K.H., Weitz C., Whittaker C., Wilming L.,
RA Wynshaw-Boris A., Yoshida K., Hasegawa Y., Kawaji H., Kohtsuki S.,
RA Hayashizaki Y.;
RT "Functional annotation of a full-length mouse cDNA collection.";